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Theory of Synaptic Plasticity in Visual Cortex*

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Abstract

A short account is given of the BCM theory of synaptic plasticity: assumptions, consequences, comparison with experiment and statistical properties. In addition a framework for comparison with other theoretical ideas is presented.

1 Introduction

Because of its great complexity, visual cortex would not seem to be an auspicious region of the brain to carry out an investigation of synaptic plasticity or of the mechanisms and sites of memory storage. It is, in addition, almost certain that much of the architecture of visual cortex is preprogrammed genetically, leaving a relatively minor percentage to be shaped or modified by experience. However the fact that visual cortex is accessible to single-cell electrophysiology, so that the output of individual cells can be measured, whereas the inputs can be controlled by varying the visual experience of the animal has made this a preferred area for experimentation and analysis. Thus over the past 30 years, a great deal of experimental and theoretical work has been done, to investigate the responses of visual cortical cells, as well as the alterations in these responses under various visual rearing conditions.

It is widely believed that much of the learning and resulting organization of visual cortex as well as other parts of the central nervous system occurs due to modification of the efficacy or strength of at least some of the synaptic junctions between neurons, thus altering the relation between presynaptic and postsynaptic potentials. The vast amount of experimental work done in visual cortex – particularly area 17 of cat and monkey – strongly indicates that one is observing a process of synaptic modification dependent on the information locally and globally available to the cortical cells. Furthermore, it is known that small but coherent modifications of large numbers of synaptic junctions can result in distributed memories. Whether and how such synaptic modification occurs, what precise forms it takes, and what the physiological and/or anatomical bases of this modification are, among the most interesting questions in this area. There is no need to assume that such mechanisms operate in exactly the same manner in all portions of the nervous system or in all

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animals. However, one would hope that certain fundamental similarities exist so that a detailed analysis of the properties of these mechanisms in one preparation would lead to some conclusions that are generally applicable.

It is our hope that such a general form of modifiability manifests itself for at least some cells of visual cortex that are accessible to experiment. If so, one then may be able to distinguish between different cortical plasticity theories with theoretical tools and the aid of sophisticated experimental paradigms. Among the difficulties faced by theoreticians are (1) adequate representation of the visual environment; (2) knowledge of what the actual inputs to cortical cells are; (3) the appropriate rule for synaptic modification; and (4) an adequate representation of the complex architecture of visual cortex.

In this article, we give a brief overview of the BCM theory of visual cortical plasticity that has been developed over the past ten years, address the difficulties mentioned above and compare the consequences of the theory with experiment. We discuss recent physiological experiments that seem to provide verification of some of the underlying assumptions of the theory, and finally, we initiate a comparison of the BCM theory with other theories that have been proposed. We assume that the reader has some familiarity with experiments demonstrating plasticity in visual cortex. A brief review may be found in Clothiaux et al., 1991.

2 BCM Theory

In what follows we give a brief overview of the BCM theory of synaptic plasticity. For a more detailed account the reader is referred to the various references cited below.

2.1 Single Cell

A typical neuron in striate cortex receives thousands of afferents from other cells. Most of these afferents derive from the lateral geniculate nucleus (LGN) and from other cortical neurons. We have approached the analysis of this complex network in several stages. In the first stage we consider a single neuron with inputs from both eyes (i.e., LGN) but without intracortical interactions.

The output of this neuron (in the linear region) can be written

$$c = m^l \cdot d^l + m^r d^r,$$

where d^l (d^r) are the LGN inputs coming from the left (right) eye to the vector of synaptic junctions m^l (m^r). The neuron firing rate (in the linear region) is therefore the sum of the inputs from the left eye multiplied by the appropriate left-eye synaptic weights plus the inputs from the right eye multiplied by the appropriate right-eye synaptic weights. Thus the neuron integrates signals from the left and right eyes. (For simplicity, whenever possible we shall omit the left and right superscripts.) According to the theory presented by Bienenstock, Cooper and Munro (BCM, 1982), the synaptic weight changes over time as a function of local and global variables: its change in time, $\dot{m}_j$, is given below:

$$\dot{m}_j = F(d_j, \ldots, m_j; d_k, \ldots, c; \bar{\bar{c}}; X, Y, Z).$$

Here variables such as $d_j, \ldots, m_j$ are designated local. These represent information (such as the incoming signal, d_j, and the strength of the synaptic junction, m_j) available locally at the synaptic junction, m_j. Variables such as $d_k, \ldots, c$ are designated quasi-local. These represent information (such as c, the firing rate of the postsynaptic cell, or d_k, the incoming signal to another synaptic

junction) that is not locally available to the junction m_j but is physically connected to the junction by the cell body itself–thus necessitating some form of internal communication between various parts of the cell and its synaptic junctions. Variables such as $\bar{\bar{c}}$ (the time averaged output of the cell) are averaged local or quasi-local variables. Global variables are designated $X, Y, Z, \ldots$. These latter represent information (e.g. presence or absence of neurotransmitters such as norepinephrine or the average activity of large numbers of cortical cells) that is present in a similar fashion for all or a large number of cortical neurons (distinguished from local or quasi-local variables presumably carrying detailed information that varies from synapse to synapse or cell to cell). Neglecting global variables, one arrives at the following form of synaptic modification equation:

$$\dot{m}_j = \phi(c, \Theta_m) d_j \tag{2.1}$$

so that the j^{th} synaptic junction, m_j, changes its value in time as the product of the input activity (the local variable d_j) and a function ϕ of quasi-local and time-averaged quasi-local variables, c and Θ_m. Θ_m is a nonlinear function of some time averaged measure of cell activity that in the original BCM formulation was proposed as

$$\Theta_m = (\bar{\bar{c}})^2. \tag{2.2}$$

In BCM, this time average is replaced, for simplicity, by a spatial average over the environmental inputs ($\bar{\bar{c}} \rightarrow m \cdot \bar{d}$). The shape of the function ϕ is given in Figure 2 for two different values of the threshold Θ_m. The occurrence of negative and positive regions for ϕ results in the cell becoming selectively responsive to subsets of stimuli in the visual environment. This happens because the response of the cell is diminished to those patterns for which the output, c, is below threshold (ϕ negative) while the response is enhanced to those patterns for which the output, c, is above threshold (ϕ positive). The non-linear variation of the threshold Θ_m with the average output of the cell contributes to the development of selectivity and the stability of the system (Bienenstock et al., 1982; Intrator and Cooper, 1992).

2.2 Cortical Network: Mean Field Theory

The actual cortical network is very complex. It includes different cell types, intracortical interactions, and recurrent collaterals. In what follows we present a method of analyzing this complex system. The first step is to divide the inputs to any cell into those from the LGN and those from all other sources. The activity of neuron i is affected by its input vector d from the LGN, and by the adjacent cortical neurons;

$$c_i = m_i \cdot d + \sum_j L_{ij} c_j, \tag{2.3}$$

where L_{ij} are the cortico-cortical synapses. Scofield and Cooper (1985, 1988) have analyzed a network extension of the single cell theory and a mean field approximation to the full network. Defining $\bar{c} = \frac{1}{N} \sum_i c_i$, where N is the number of neurons in the network, the mean field approximation is obtained by replacing the inhibitory contribution of cell j, c_j by its average value so that c_i becomes:

$$c_i = m_i \cdot d + \bar{c} \sum_j L_{ij}. \tag{2.4}$$

From a consistency condition it follows that $\bar{c} = \bar{m} \cdot d + \bar{c} L_0 = (1 - L_0)^{-1} \bar{m} \cdot d$, where $\bar{m} = \frac{1}{N} \sum_i m_i$, and $L_0 = \frac{1}{N} \sum_{ij} L_{ij}$, so that $c_i = (m_i + (1 - L_0)^{-1} \bar{m} \sum_j L_{ij}) d$.

If we assume that the lateral connection strengths are a function only of the relative distance $i - j$, then L_{ij} becomes a circular matrix so that $\sum_i L_{ij} = \sum_j L_{ij} = L_0$, and

$$c_i = (m_i + L_0(1 - L_0)^{-1} \bar{m}) d. \tag{2.5}$$

In the mean field approximation, one can therefore write $c_i(\alpha) = (m_i - \alpha) d$, with $\alpha = |L_0|(1 + |L_0|)^{-1} \bar{m}$.

When analyzing the position and stability of the fixed points using this approximation, it follows under some mild assumption on the evolution of the average synaptic weights, that there is a mapping

$$m_i' \leftrightarrow m_i(\alpha) - \alpha$$

such that for every neuron in such a network with synaptic weight vector m_i there is a corresponding neuron with weight vector m_i' that undergoes the same evolution (around the fixed points) subject to a translation α.

Although the averaged inhibition assumption used in the mean-field theory is an approximation, the mean field network described above provides a powerful tool to analyze a certain type of network architecture in great detail, and to gain an intuitive understanding of a complex network in terms of the behavior of a single neuron.

2.3 Synapses with Varying Modifiability

In the equations above, all synapses are taken to be modifiable in the same way. However, the behavior of visual cortical cells in various rearing conditions suggests that some cells respond more rapidly to environmental changes than others. In monocular deprivation, for example, some cells remain responsive to the closed eye in spite of the very large shift of most cells to the open eye. Hubel and Wiesel (1959) and Singer (1977) , found, using intracellular recording, that geniculo-cortical synapses on inhibitory interneurons are more resistant to monocular deprivation than are synapses on pyramidal cell dendrites. These results suggest that some LGN-cortical synapses modify rapidly, while others modify relatively slowly, with slow modification of some cortico-cortical synapses. Excitatory LGN-cortical synapses onto excitatory cells may be those that modify primarily. Since these synapses are formed exclusively on dendritic spines, this raises the possibility that the mechanisms underlying synaptic modification exist primarily in axo-spinous synapses. To embody these facts we introduce two types of LGN-cortical synapses: those (m_i) that modify according to the modification rule discussed in BCM and those (z_k) that remain relatively constant. In a cortical network with modifiable and non- modifiable LGN-cortical synapses, and non-modifiable cortico-cortical synapses L_{ij}, the synaptic evolution equations become

$$\begin{aligned} \dot{m}_i &= \phi(c_i, \Theta_m{}^i) d, \\ \dot{z}_k &= 0, \\ \dot{L}_{ij} &= 0. \end{aligned} \tag{2.6}$$

As will be discussed below, such a network is capable of explaining the variety of experiments considered.

3 BCM and the Neurobiology of Synaptic Modification

The BCM theory and its recent extensions originated as an attempt to account for the varied consequences of different visual environments on the developing visual cortex. In cats, the circuitry of the visual cortex can be modified by simple manipulations of visual experience during a "critical period" in the first few months of postnatal development. For example, one such manipulation, monocular deprivation, leads to a disconnection of the inputs from the deprived eye which renders the animal behaviorally blind through that eye. The goal of the BCM theory is to develop a model of synaptic modification which accounts for those striking changes in visual cortex that result from alterations in the patterns and amount of activity arising at the two retinae. While the theory aims to provide a physiologically-plausible account of synaptic plasticity, it does not address the mechanism by which plasticity diminishes at the end of the critical period. A number of possible mechanisms have been proposed to account for the short duration of the plastic period but at present it is not clear that the length of the critical period is determined by the same mechanism as that underlying synaptic change.

The validity of the BCM theory, as with any theory, can be tested in two ways. The first is to derive predictions or consequences of the theory in various situations that can be compared with experimental results. There is a considerable experimental literature on visual cortical plasticity reaching back 30 years which facilitates such comparisons with the BCM theory. The second approach is to attempt to verify the underlying assumptions of the theory, particularly those assumptions that distinguish it from others. In the case of BCM the most important and unique assumptions concern the form of the synaptic modification function ϕ and the movement of the modification threshold. Over the last five years we have made significant progress using both of these approaches, and this work is summarized briefly below.

3.1 Comparison of Theory and Experiment

In work recently published by Eugene Clothiaux and colleagues (Clothiaux et al., 1991) the consequences of the BCM theory were compared in detail with the results of experiments on what were called "classical" rearing conditions. These conditions include normal binocular vision, monocular deprivation, reverse suture, strabismus, binocular deprivation, as well as the restoration of normal binocular vision after various forms of deprivation. Comparisons with the pharmacological manipulations that affect visual cortical plasticity (e.g. Greuel et al., 1987; Reiter and Stryker, 1988; Bear et al., 1990) were not considered and remain an area that is ripe for further work. The modifications considered by Clothiaux et al. were those that occur in kitten visual cortex during the second postnatal month after brief ($\simeq$ 2 weeks) changes in visual experience. Particular attention was given to the manner in which the theory predicts that changes in visual experience should affect the binocularity of cortical neurons and the selectivity of these neurons for the stimulus pattern (eg. its orientation). It is these properties of binocularity and selectivity which distinguish cortical neurons from those in the retina and thalamus. A review of the experimental literature as it relates to the modification of these properties may be found in Clothiaux et al. (1991).

All theories of visual cortical plasticity have to make some assumption as to how the initial visual scenes are converted into LGN firing rates and how this information reaches visual cortex. We wish to model the input to visual cortex that arises from the regions of the two retinae that view the same point in visual space. For simplicity, Clothiaux et al. assumed that LGN activity is a direct reflection of retinal ganglion cell activity. Two types of LGN-cortical input were modeled: (1)

activity elicited when visual contours are presented to the retinae, which we call "pattern" input; and (2) activity that arises in the absence of visual contours, which we call "noise". From our point of view the important distinction between pattern and noise input is the degree of correlation that the two types of input produce in the LGN. For a specific input "pattern" the activity of one LGN neuron is assumed to have a predictable relationship (i.e. correlation) to the activity of other LGN neurons, while for "noise" the activity of one LGN neuron is independent of the activity of the other LGN neurons[1]. Differences between distinct patterns (for example, between various stimulus orientations) are reflected by the differences in their distribution of activity across the LGN. Using this type of pattern input distorted by noise, and noise alone, Clothiaux et al. were able to reproduce both the outcome and kinetics of synaptic change in visual cortex resulting from normal visual experience and a wide variety of visual deprivation conditions.

As one example of the quantitative nature of these results, consider the simulation of the effects of monocular deprivation (Figure 7, Clothiaux et al., 1991). Beginning from a state in which the simulated neuron is binocularly responsive and selective, substituting pattern input through one eye with noise leads to a rapid synaptic disconnection of the "deprived" eye. Mathematical analysis provides a complete account of the factors on which this result depends if it is governed by the principles of the BCM theory. For example, for this result to be obtained using BCM it is necessary that the neuron be selective (i.e. that it responds vigorously only to a fraction of the patterns that are presented to the "open eye") before the ocular dominance changes, and that the deprived eye inputs carry noise (i.e. that they be active). The prediction that the ocular dominance shift depends on neuronal selectivity was tested by Paradiso and colleagues (Ramoa et al., 1988). They found that cortical infusion of the GABA receptor antagonist bicuculline, which greatly reduces orientation selectivity in visual cortex, eliminates the ocular dominance shift that normally results from monocular deprivation. The second prediction that the disconnection of the deprived eye depends on noise has never been tested explicitly, but there are some indications that it is also correct. For example, clinical observations in humans led Jampolsky (1978) to conclude that the effects of monocular diffusion (resulting from lid suture) are more severe than the effects of monocular occlusion (resulting from an opaque eye-patch or contact lens).

To determine the time equivalence of each iteration for the parameters used, the behavior of the model under monocular deprivation can be compared to the results of the corresponding experiment. Equivalence was established between the number of computer iterations and the duration of deprivation required for complete disconnection of the deprived eye (Clothiaux et al. 1991). Thus, using a fixed set of parameters, one has a direct correspondence between the temporal dynamics of synaptic change in the theory and experiments. This can be used to analyze and compare kinetics and outcome of theory and experiment for other manipulations. For example, in "reverse suture", the deprived eye is opened and the open eye is closed after a period of initial monocular deprivation. Experimentally, it is observed that the newly closed eye shows a greatly reduced response in about 24 hours, but that the recovery of the response to the newly open eye generally does not begin for another 1-2 days (Mioche and Singer, 1989). The same difference in the time required to obtain the initial effect and the reversal is seen with the model. The correspondence of theory and experiment is thus very close. The theoretical explanation for this result is that recovery requires that the modification threshold slide nearly to zero and, using the same parameters that were fixed for monocular deprivation, this requires approximately 24 hours.

[1] Addition of local correlations such as those suggested by the work of Mastronarde (1989) does not alter the results.

Similar comparisons for the other experimental manipulations are discussed in detail in Clothiaux et al. (1991). We conclude that when the predictions of the theory have been tested they are in good agreement with what is seen experimentally.

3.2 Neurobiological Foundations for the Assumptions of the BCM Theory

Recent advances in our understanding of excitatory amino acid (EAA) receptors have suggested a possible physiological basis of the BCM form of synaptic modification. In 1987, Bear et al. proposed that the modification threshold Θ_m of BCM related to the membrane potential at which the N-methyl-D-aspartate (NMDA) receptor dependent Ca^{2+} flux reached the threshold for inducing synaptic long-term potentiation (LTP). In support of the hypothesis that NMDA receptor mechanisms play a role in synaptic plasticity, Bear and co-workers have found that the pharmacological blockade of NMDA receptors with the competitive antagonist AP5 disrupts the physiological (Kleinschmidt et al., 1987; Bear et al., 1990) and anatomical (Bear and Colman, 1990) consequences of monocular deprivation in striate cortex. Although the interpretation of these experiments is compromised by the finding that AP5 reduces visually evoked responses (Fox et al., 1989), the data indicate that activity evoked in visual cortex in the absence of NMDA receptor activation is not sufficient to produce loss of closed-eye responsiveness in MD.

In the past several years our work has been focused on the synaptic plasticity that can be evoked in brain slices to better investigate the assumptions of the BCM theory and to address possible underlying mechanisms (Connors and Bear, 1988; Press and Bear, 1990; Bear et al., 1992; Dudek and Bear, 1992; Kirkwood et al., 1992). Hippocampus, particularly CA1 and dentate gyrus, is an advantageous preparation because robust and long-lasting experience-dependent synaptic modifications can be evoked in this structure. Serena Dudek in Bear's lab (1992) recently tested a theoretical prediction that patterns of excitatory input activity that consistently fail to activate target neurons sufficiently to induce synaptic potentiation will instead cause a specific synaptic depression. To realize this situation experimentally, the Schaffer collateral projection to CA1 in rat hippocampal slices was stimulated electrically at frequencies ranging from 0.5 to 50 Hz. 900 pulses at 1-3 Hz consistently yielded a depression of the CA1 population EPSP that persisted without signs of recovery for > 1 hour following cessation of the conditioning stimulation. This long-term depression was specific to the conditioned input and could be prevented by application of NMDA receptor antagonists. This result was surprising in that NMDA receptors are known to participate in the induction of long-term potentiation, an increase in synaptic effectiveness. Indeed, at higher stimulation frequencies the depression was replaced by a potentiation. If the effects of varying stimulation frequency in the experiments of Dudek and Bear are explained by different values of postsynaptic response (perhaps the integrated postsynaptic depolarization or Ca^{2+} level) during the conditioning stimulation, then it can be seen from Figure 3 that their data are in striking agreement with assumptions of the BCM theory.

Of course, as striking as this similarity is, Dudek's work was performed in hippocampus and the BCM theory was developed for visual cortex. And, although these two forms of synaptic plasticity (depression and potentiation) have been reported in the sensory neocortex (cf. Artola et al., 1990), evidence to date has indicated that they occur with far lower probability, usually require pharmacological treatments for their induction, and are elicited by stimulation patterns that differ dramatically from those that are effective in hippocampus (see discussion in Bear et al., 1992). Together, these data have been taken as support for the view that hippocampus and sensory

neocortex may be quite distinct with respect to their capability for synaptic change. However, a direct comparison of plasticity of synaptic responses evoked in adult rat hippocampal field CA1 with those evoked in adult rat and immature cat visual cortical layer III has now been carried out by Alfredo Kirkwood and colleagues in Bear's lab (1992). In the neocortical preparations they have stimulated the direct input to layer III from layer IV rather than using the traditional approach of stimulating the white matter, and find, contrary to the prevailing view, that very similar forms of plasticity, LTP and LTD, are evoked with precisely the same types of stimulation in the three types of cortex without the use of pharmacological treatments. Further, in all three preparations, both LTP and LTD depend on activation of NMDA receptors. These data suggest, first, that hippocampus should not be considered as a privileged site for plasticity in the adult brain and, second, that a common principle may govern experience-dependent synaptic plasticity, both in CA1 and throughout the superficial layers of the neocortex. We believe that this work represents an important advance towards a general theory of experience-dependent synaptic plasticity in the mammalian brain.

It is our opinion that in its entirety this work gives strong justification for a form of modification similar to that assumed by BCM. However, still open is the question of the sliding modification threshold. Although more work remains to be done on this question, we note that two recent studies have shown that the sign and magnitude of a synaptic modification in both hippocampus (Huang et al., 1992) and the Mauthner cell of goldfish (Yang and Faber, 1991) have been shown to depend on the recent history of synaptic activation.

4 Reformulation and Extensions of the BCM Theory

In order to compare the BCM theory with other theories of synaptic plasticity as well as to exhibit its information processing and statistical properties, the following formulation proves convenient.

4.1 Objective function formulation

In a recent statistical formulation of the BCM theory (Intrator and Cooper, 1992), the threshold Θ_m was defined[2] as

$$\Theta_m = E[(x \cdot m)^2],$$

and an energy function that corresponds to a risk function in statistical decision theory was presented:

$$R_m = -\mu\{\frac{1}{3}E[(x \cdot m)^3] - \frac{1}{4}E^2[(x \cdot m)^2]\}. \tag{4.1}$$

It was shown that the differential equations describing synaptic weight modification are a stochastic approximation of the negative gradient of the risk, hence tending to minimize this risk (Intrator and Cooper, 1992, for review). This formulation permits us to demonstrate the connection between the unsupervised BCM learning procedure and various statistical methods, in particular, that of Exploratory Projection Pursuit (Friedman, 1987). It also provides a general method for stability analysis of the fixed points of the theory and enables us to analyze the behavior and the evolution of the network under various visual rearing conditions. In the next few sections we shall use this

[2]To use the same notation as in Intrator and Cooper (1992), we denote the input as the vector x. This is equivalent to d used above.

formulation to extend the theory to nonlinear neurons, and consequently to a network of feedforward inhibitory neurons.

4.2 Nonlinear Neurons

From statistical considerations that are motivated by the projection pursuit ideas, it is more effective to consider a nonlinear neuron that is less sensitive to possible outliers in the data. This is done by defining the neuron's activity as $c = \sigma(x \cdot m)$, where σ usually represents a smooth sigmoidal function. It is also desirable to have the ability to shift the projected distribution (of the input data) so that one of its peaks is at zero, by introducing a threshold β so that the projection is defined to be $c = \sigma(x \cdot m + \beta)$. From the biological viewpoint, β can be considered as spontaneous activity. The modification equations for finding the optimal threshold β are easily obtained by observing that this threshold effectively adds one dimension to the input vector and the vector of synaptic weights so that $x = (x_1 \ldots, x_n, 1)$, $m = (m_1, \ldots, m_n, \beta)$, and therefore, β can be found by using the same synaptic modification equations. For the rest of the paper we shall assume that this threshold is added to the projection, without specifically writing it.

For the nonlinear neuron, Θ_m is defined to be $\Theta_m = E[\sigma^2(x \cdot m)]$. The gradient of the risk becomes:

$$-\nabla_m R_m = \mu \; E[\phi\Big(\sigma(x \cdot m), \Theta_m\Big)\sigma' x], \tag{4.2}$$

where σ' represents the derivative of σ at the point $(x \cdot m)$. Note that the multiplication by σ' reduces sensitivity to outliers of the differential equation since for outliers σ' is close to zero. The gradient decent procedure is valid, provided that the risk is bounded from below (cf. Intrator and Cooper, 1992).

4.3 Networks with Feed-Forward Inhibition: Application to Classification

Intrator and Cooper (1992) have extended the single cell theory to a feed forward inhibition network which does not require the mean field approximation; nor does it require that the cortico-cortical synapses L_{ij} be constant. Thus it is possible to study networks with varying amounts of excitation and inhibition.

The activity of neuron k in the network is $c_k = x \cdot m_k$, where m_k is the synaptic weight vector of neuron k. The *inhibited* activity and threshold of the k'th neuron are given by

$$\tilde{c}_k = c_k - \eta \sum_{j \neq k} c_j, \qquad \tilde{\Theta}_m^k = E[\tilde{c}_k^2]. \tag{4.3}$$

The relation between the feed forward inhibition network and the mean field network is discussed in Intrator and Cooper (1992).

For the feed-forward network the risk for node k is given by:

$$R_k = -\mu\{\frac{1}{3}E[\tilde{c}_k^3] - \frac{1}{4}E^2[\tilde{c}_k^2]\}, \tag{4.4}$$

and the total risk is given by

$$R = \sum_{k=1}^{N} R_k. \tag{4.5}$$

It follows that the gradient of R becomes:

$$\begin{aligned}\frac{\partial R}{\partial m_k} &= \frac{\partial R_k}{\partial m_k} - \eta \sum_{j \neq k} \frac{\partial R_j}{\partial m_j} \\ &= \mu \left[E[\phi(\tilde{c}_k, \tilde{\Theta}_m^k)x] - \eta \sum_{j \neq k} E[\phi(\tilde{c}_j, \tilde{\Theta}_m^j)x] \right]. \end{aligned} \tag{4.6}$$

The equation performs a constraint minimization in which the derivative with regard to one neuron can become orthogonal (when $\eta \rightarrow 1$) to the sum over the derivatives of all other synaptic weights. Nevertheless, the coupling between the neurons is very simple to calculate and does not require any matrix inversion. Equation 4.6 therefore, allows a simple computational algorithm that performs exploratory projection pursuit of several projections in parallel.

When the nonlinearity of the neuron is included, the inhibited activity is defined (as in the single neuron case) as $\tilde{c}_k = \sigma(c_k - \eta \sum_{l \neq k} c_l)$. $\tilde{\Theta}_m^k$, and R_k are defined as before. However, in this case

$$\frac{\partial \tilde{c}_k}{\partial m_j} = -\eta \sigma'(\tilde{c}_k)x, \quad \frac{\partial \tilde{c}_k}{\partial m_k} = \sigma'(\tilde{c}_k)x. \tag{4.7}$$

Therefore the total gradient becomes:

$$\dot{m}_k = \frac{\partial R}{\partial m_k} = \mu \{ E[\phi(\tilde{c}_k, \tilde{\Theta}_m^k)\sigma'(\tilde{c}_k)x] - \eta \sum_{j \neq k} E[\phi(\tilde{c}_j, \tilde{\Theta}_m^j)\sigma'(\tilde{c}_j)x] \}. \tag{4.8}$$

This biologically motivated system of equations has many desirable statistical properties and has been applied to various non-trivial feature extraction tasks such as phoneme recognition (Intrator, 1992) and 3D object recognition (Intrator and Gold, 1993).

5 Comparison of BCM with Other Visual Cortical Plasticity Theories

In order to compare ideas concerning visual cortical plasticity, it is important to analyze separately the different components that make up a theory, and to compare theories feature by feature. We consider a theory as being composed of the following three components:

- Synaptic modification equations.
- Model of the input environment.
- Network architecture.

In some cases, there are interactions between these components that are not explicitly defined. For example, several theories are said to have the property of being able to develop orientation selectivity prenataly, i.e., using random noise as an input environment (Linsker, 1986; Miller et al., 1989). However, under closer examination, it turns out that they have architectual constraints that actually yield very different input environments. Inputs to the network become strongly locally correlated after the first layer due only to network architecture (the arborization function). The arborization function determines the density of synapses as a function of planar distance from their target cell. This correlated input can then drive the higher level of cells to develop orientation selective cells. When the arborization function is uniform, all the weights of all layers will become positively saturated thus no selectivity will develop.

5.1 Comparison Based on Synaptic Modification Equations

In order to examine the effect of the synaptic modification equations in isolation, we shall fix the network inputs to be the same, and fix the architecture as well. The simplest architecture that would already yield a significant difference between several models would be of a single cortical neuron receiving input from a single source (single eye).

In the correlation of activity models (Sejnowski, 1977; Linsker, 1986; Kammen and Yuille, 1988; Yuille et al., 1989; Miller et al., 1989) the input is defined in terms of the correlation of activity in the presynaptic afferents, whereas in the BCM model the input is defined in terms of the presynaptic activity. For reasons that will become clear, it is difficult to transform the correlation of activity models to a presynaptic activity models; however, we can rewrite the BCM model as a correlation of activity model.

To simplify notation and without loss of generality we shall assume that the input activity in each ganglion cell has zero mean. First we show how the transformation from input activity to correlation activity is done by expanding on footnote 15 of Miller et al. (1989). This will be done in the simple case of a single cortical neuron with no interaction between LGN inputs coming from the two eyes. Miller's rule has the following form:

$$\frac{dS(\alpha,t)}{dt} = \lambda A(-\alpha)[c(t) - c_1]a_\sigma(\alpha,t) - \gamma S(\alpha,t) - \epsilon' A(-\alpha), \tag{5.1}$$

where α is the afferent location, A is the arbor function representing the number (or in the limit, the density of) afferents coming from location α, $a_\sigma(\alpha,t)$ is the afferent activity at location α, the subscript σ represents an addition of threshold and saturation effects, and c_1 is a constant. $c(t)$ is the neuronal activity which in this simple case of no lateral interactions is given by $c(t) = \sum_\beta S(\beta,t)a_\sigma(\beta,t) + c_2$ where c_2 is some constant. $\gamma S(\alpha,t) + \epsilon' A(-\alpha)$ are decay functions of the synaptic weights. Substituting $c(t)$ into 5.1, denoting $c_3 = c_2 - c_1$ and taking the average over the input space (at a given afferent location) we obtain

$$\frac{dS(\alpha,t)}{dt} = \lambda A(-\alpha)\{\sum_\beta S(\beta,t)E(a_\sigma(\beta,t)a_\sigma(\alpha,t)) + c_3 E(a_\sigma(\alpha,t))\} - \gamma S(\alpha,t) - \epsilon' A(-\alpha). \tag{5.2}$$

Using $C(\alpha,\beta)$ to represent the correlation of activity $C(\alpha,\beta) \stackrel{\text{def}}{=} E(a_\sigma(\alpha,t)a_\sigma(\beta,t))$ we get

$$\frac{dS(\alpha,t)}{dt} = \lambda A(-\alpha)\{\sum_\beta S(\beta,t)C(\alpha,\beta) - c_1 E[a_\sigma(\alpha,t)]\} - \gamma S(\alpha,t) - \epsilon' A(-\alpha), \tag{5.3}$$

which is a simple case of equation (1) in Miller et al. (1989).

Analogous reformulation is done below for the BCM modification equation

$$\frac{dm(\alpha,t)}{dt} = \lambda\phi(c(t),\Theta_m)a(\alpha,t), \tag{5.4}$$

for the synaptic weight m (in Miller's notation this is S): for simplicity we omit decay terms and assume a uniform arbor function.

Using a simple form of the modification function, $\phi(c,\Theta_m) \stackrel{\text{def}}{=} c(c - \Theta_m)$ (Bienenstock et al., 1982) substituting $c(t) = \sum_\beta m(\beta)a(\beta,t)$ and taking the average over the input space at a given

afferent location we obtain

$$\frac{dm(\alpha,t)}{dt} = \lambda E\{a(\alpha)\sum_{\beta\gamma} m(\beta)a(\beta)m(\gamma)a(\gamma)\} - E\{a(\alpha)\sum_{\beta} m(\beta)a(\beta)\}\Theta_m. \tag{5.5}$$

Wherever it is clear we omit the dependency of $a(\alpha)$ on t (assuming it is a stationary process). Using the same correlation function as defined above, and defining the third order correlation $\tilde{C}(\alpha,\beta,\gamma)$ of the input activity to be $\tilde{C}(\alpha,\beta,\gamma) = E[a(\alpha)a(\beta)a(\gamma)]$, yields

$$\frac{dm(\alpha,t)}{dt} = \lambda\{\sum_{\beta,\gamma} \tilde{C}(\alpha,\beta,\gamma)m(\beta)m(\gamma) - \Theta_m \sum_{\beta} m(\beta)C(\alpha,\beta)\}. \tag{5.6}$$

Using a definition for the threshold $\Theta_m \stackrel{\text{def}}{=} E[\sum_\alpha a(\alpha)m(\alpha)]^2$ (Intrator, 1990; Intrator and Cooper, 1992) and using the second order correlation function C, Θ_m becomes $\Theta_m = \sum_{\gamma,\delta} C(\gamma,\delta)m(\gamma)m(\delta)$. Therefore, eq. (5.6) becomes

$$\frac{dm(\alpha,t)}{dt} = \lambda\{\sum_{\beta,\gamma} \tilde{C}(\alpha,\beta,\gamma)m(\beta)m(\gamma) - \sum_{\beta\gamma\delta} C(\alpha,\beta)C(\gamma,\delta)m(\beta)m(\gamma)m(\delta)\}. \tag{5.7}$$

A few important observations follow. The correlation based models use only the first and second order statistical information[3] of the data, whereas BCM utilizes in addition the third order statistics of the input activity. Therefore, without going into the details of the limiting behavior, this already suggests that correlation based models are less sensitive to the input environment. Analysis shows that in many cases the correlation of activity models find the principal components of the input environment (Oja, 1982; Kohonen, 1984; Yuille et al., 1989; Miller et al., 1989; Sanger, 1989; Granger et al., 1989). In the following section we discuss some properties of principal components in information processing.

5.1.1 Comparison Based on the Information Extraction Properties

It now becomes relevant to ask what type of structure can be extracted from first and second moments only, and what constitutes an *interesting structure*. The first question is quite old and its answer is well known; First and second moments contain information about the principal components of the input distribution, which are those directions that can minimize L^2 error between the original data and the reconstructed data based only on the first few leading components. Another way to view principal components is to observe that they maximize the variance of the projected distribution, namely the variance of the new random variable that is the projection of the inputs onto the principal components.

Principal Components and Maximum Information Preservation

Networks that extract principal components from data are numerous, e.g., (Sejnowski, 1977; Oja, 1982; Linsker, 1986; Kammen and Yuille, 1988; Yuille et al., 1989; Miller et al., 1989; Sanger, 1989; Granger et al., 1989). Linsker presented the principles guiding synaptic modification in his layered network and showed that the development rule causes a cell to develop so as to maximize

[3] In other words, the mean and covariance matrix of input activity

the variance of its output activity, subject to the constraint on the total connection strength, and on each synaptic value (Linsker, 1988). Linsker then describes the connection of this rule to principal component analysis and to the principle of *maximum information preservation* taken from information theory[4]. This principle is optimal when the goal is to accurately reconstruct the input, but is not optimal when the goal is classification. This is shown in the following simple example (Figure 1, see also p. 212 Duda and Hart, 1973). Two clusters each belonging to a different class are presented. The goal is to find a single dimensional projection that will capture the structure information in the data. In Figure 1 (B) different clusters have different variance in either direction, whereas in Figure 1 (A) the variance in both directions is equal. Clearly, the structure in the data is conveyed in the x projection however, in the first example the variance is maximized in the y projection. This projection also minimizes the mean squared error (MSE) and is therefore superior from maximum information preservation. In the second example, due to the fact that the variance of each cluster is equal in both directions, the optimal projection that capture the most structure in the data, and preserves maximum information is the x projection.

Another way to view what principal components do to the data is by observing that they define a new system of coordinates in which the covariance matrix is diagonal, namely, they eliminate the second order correlation in the data i.e., correlation between the projections of the input data onto any two principal components. It is important to note here that this procedure does not eliminate higher order correlation in the data.

Finding Other Interesting Low-Dimensional Structure in Data

This problem has recently been discussed in the context of a statistical method called projection pursuit (PP) (Huber, 1985, for review). This method seeks structure that is exhibited by (linear) projections of the data, and is therefore relevant to neural network theory since the activity of a neuron is believed to be a function of the projection of the inputs on the vector of synaptic weights. Diaconis and Freedman (1984), have shown that for most high-dimensional clouds, most low-dimensional projections are approximately normal. This finding suggests that important information in the data is conveyed in those directions whose single dimensional projected distribution is far from Gaussian. For example, some known measures of deviation from normality are skewness and kurtosis which are functions of the first four moments of the distribution. These moments contain information about statistical correlations up to fourth order. Intrator (1990) has shown that a BCM neuron (given by equation 4.8) can find structure in the input distribution that exhibits deviation from normality in the form of multi-modality in the projected distributions. This type of deviation, which is measured by the first three moments of the distribution, is particularly useful for finding clusters in high dimensional data (since clusters can not be found directly in the data due to its sparsity) and is thus useful for classification or recognition tasks. Below, we give another interpretation of this projection index in light of the previous discussion.

If we assume that the retina is performing decorrelation of the inputs (Atick and Redlich, 1992) then the covariance matrix $C(\alpha,\beta)$ is diagonal (assuming that the inputs have zero mean) and so for eigen values $e(\alpha)$, equation 5.7 becomes:

$$\frac{dm(\alpha,t)}{dt} = \lambda\{\sum_{\beta,\gamma} \tilde{C}(\alpha,\beta,\gamma)m(\beta)m(\gamma) - e(\alpha)m(\alpha)\sum_{\gamma} e(\gamma)m^2(\gamma)\}. \tag{5.8}$$

[4]For review in this context see (Linsker, 1988).

This suggests that the BCM synaptic modification equation is performing third order decorrelation of the inputs subject to some penalty related to the size of the weights. When the second order statistics of the input data is not decorrelated, then the modification equation can be thought of trying to find some balance between third order correlation and second order correlation in the data.

5.2 Comparison Based on Assumptions About the Input Environment

We summarize in Table 1 the different assumptions about the environment used by our group and by Miller and colleagues to model classical visual deprivation experiments. What is apparent from this comparison is the different emphasis given to between-eye and within-eye correlations in activity. To quote Mastronarde: "Some of the strongest evidence on the importance of correlated firing in development comes from cases where local correlations in activity are induced by sensory stimulation; e.g. formation of binocular cells in visual cortex requires binocularly corresponding visual input to the two eyes (Hubel and Wiesel, 1965). There has been growing interest in a more restricted question: what is the role in development of the correlated activity that occurs in the spontaneous discharge?" (Mastronarde, 1989). In our work we have chosen to focus on the influence of activity induced by sensory stimulation on the development of visual cortex.

5.3 Comparison Based on Network Architecture

Although it is possible that network architecture plays an important role in comparison of various ideas on visual cortical plasticity, in this chapter we have not done any analysis of different architecture. This will be dealt with in subsequent work.

6 Concluding Remarks

We have given a short account of the BCM theory of synaptic plasticity – comparison with experiments in visual cortex and possible cellular and molecular basis for the fundamental modification equations. In addition we have shown that correlation based models and BCM differ in the type of structure for which they search; Correlation models include first and second order statistics of input correlations, while BCM modification also includes third order statistics.

Evidence exists for a principal component type preprocessing that may be taking place in the retina; we suggest that BCM modification further preprocess the visual inputs by reducing (extracting) third order statistical correlations. Extracting third order statistics from the visual environment is a natural extension and complements the extraction of second order statistics that may be done in the retina.

Statistical theory tells us that finite order statistics of the data is not sufficient to uniquely characterize the data distribution. However, the addition of the third order moment adds important feature such as skewness to the description of the distribution (Kendall and Stuart, 1977, for review), and in this case, it adds information about multimodality (Intrator, 1992). The method of principal components is sufficient for finding clusters in data when the variance of each cluster is relatively constant in all directions, since then directions that maximize the variance also maximize the information conveyed for the purpose of classification. When this is not the case, the example (Figure 1) shows that the direction that maximizes the variance of the projection does not necessarily carry information useful for separation of the two clusters although it does find the direction

that will minimize the mean squared error between the reconstructed signal and the input; this is dictated by the principle of maximum information preservation.

It is possible that the principle of maximum information preservation is useful in retinal processing, in which an order of magnitude reduction in the number of cells occurs. We suggest that this principle is not general enough to account for processing done in early visual cortex. and that such statistical properties provide a convenient framework for comparison of various plasticity theories.

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Table Caption

Table 1 Summary of the different assumptions about the input environment between Clothiaux et al. (1992) and Miller et al. (1989). (Normal Rearing (NR), Monocular Deprivation (MD), and Strabismus (ST)).

Figure Captions

Figure 1 Principal components find useful structure in data (A) and fail when the variance of each cluster is different in each direction (B).

Figure 2 The ϕ function for two different Θ_m's.

Figure 3 Comparison of experimental observations with BCM ϕ function for synaptic modification. Data replotted from Dudek and Bear (1992).

	Clothiaux et al. (1991)	Miller et al. (1989)
NR	• Patterned input (correlated activity within the eye with addition of noise) from both eyes. • Correlation between eyes.	• Locally correlated input from both eyes. • No correlation between eyes.
MD	• Patterned input from the open eye. • Uncorrelated noise from the deprived eye.	• Same correlation structure as NR. • Reduced activity to the deprived eye.
ST	• Patterned input from each eye. • No correlation between eyes.	• Locally correlated input from both eyes. • Anti correlations between eyes.

Table 1:

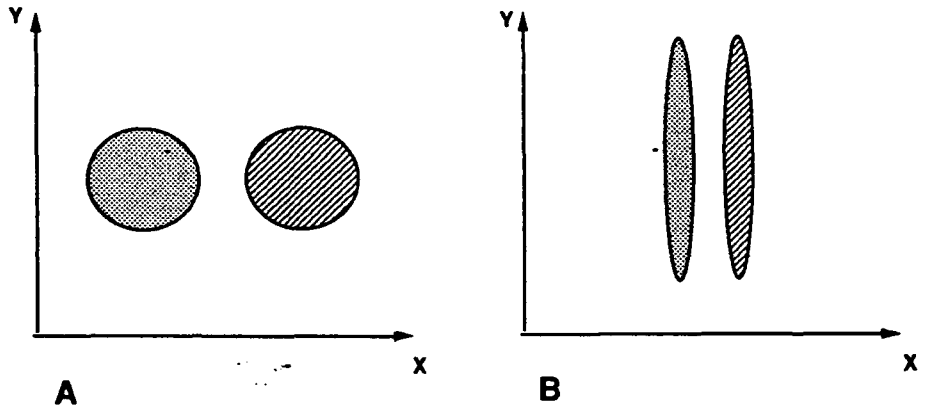

Figure 1:

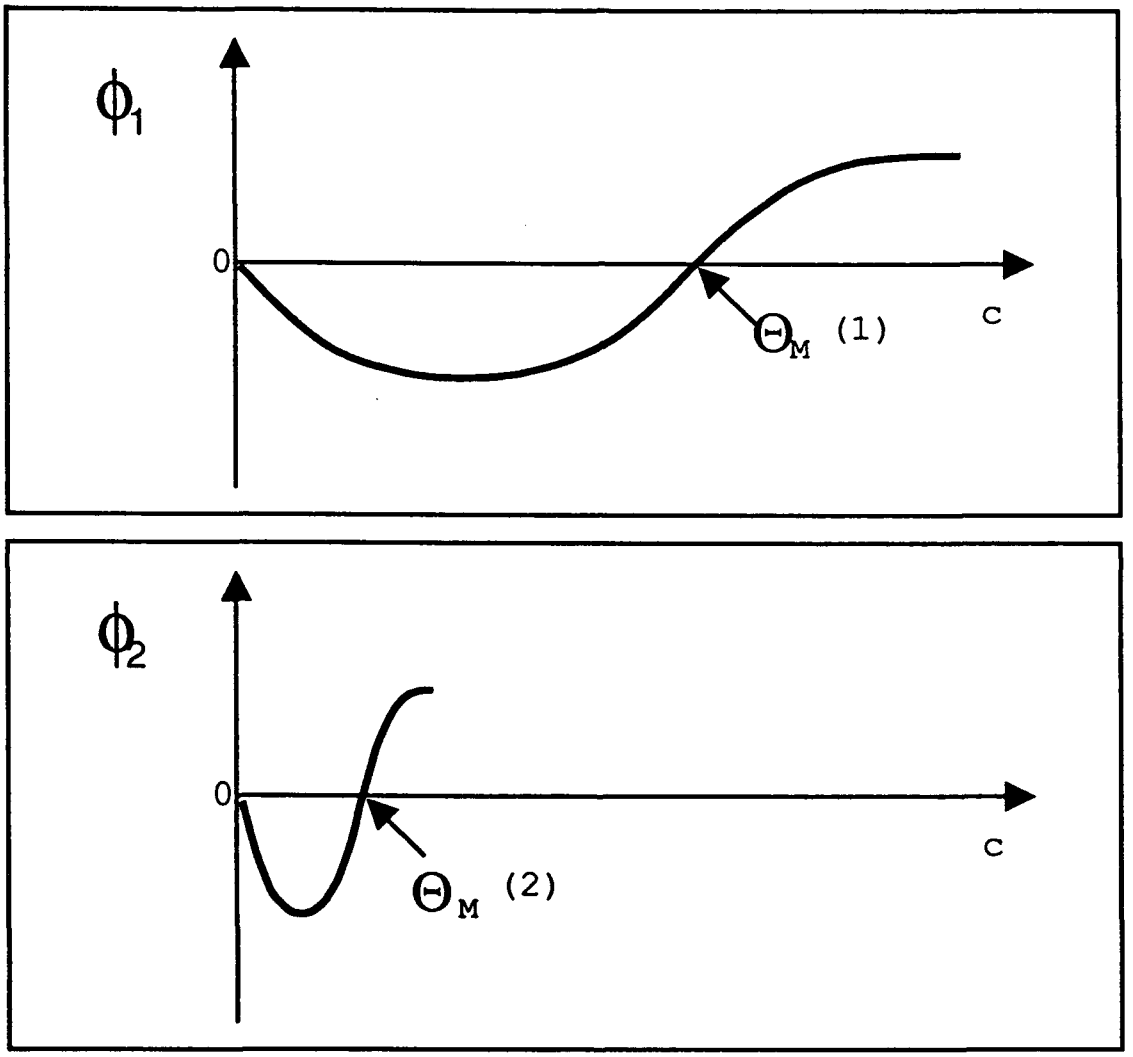

Figure 2:

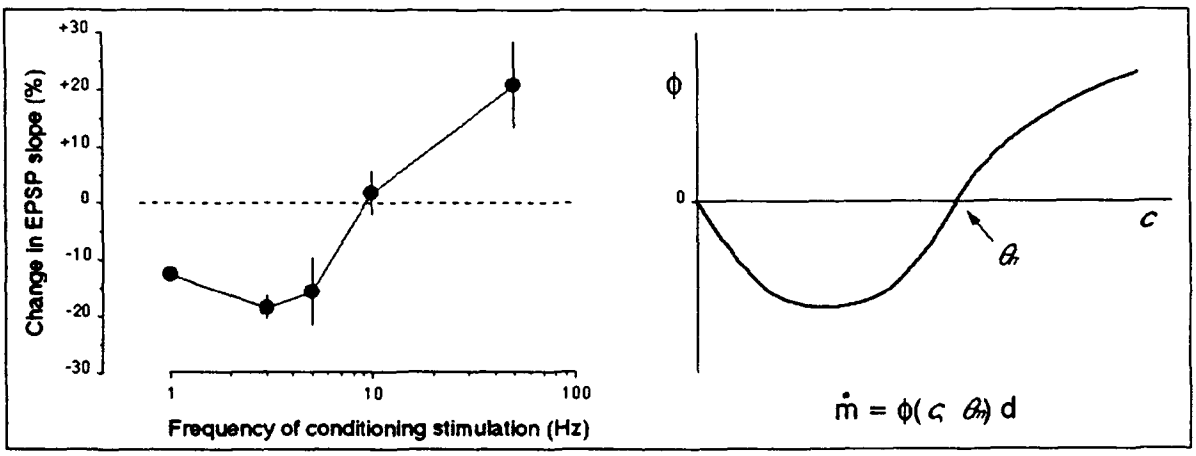
+30
+20
+10
0
-10
-20
-30
Change in EPSP slope (%)
1
10
100
Frequency of conditioning stimulation (Hz)
ϕ
0
c
θm
ṁ = ϕ(c θm) d

Figure 3: